## **AMENDMENTS TO THE SPECIFICATION:**

Please amend the specification as follows:

Please replace the paragraph on page 1, last full paragraph towards the bottom, beginning with "[t]o make a diagnosis or a ...," with the following paragraph.

To make a diagnosis <u>Diagnosis</u> or a post-treatment diagnosis <u>for of leukemia</u>, preleukemia or aleukemic malignant blood diseases <u>are is</u> important for determining the strategy for treating these diseases.

Please replace the paragraph beginning on page 1, last sentence at the bottom, beginning with "[f]or diagnosing leukemia at its first ...," and continuing onto page 2, with the following paragraph.

For diagnosing leukemia at its first occurrence, there is a method in which the white blood cell count in the peripheral blood of a patient is determined, and when When the white blood cell count is beyond the normal level, the occurrence of leukemia is suspected. However, the white blood cell counts also increase due to the enhancement of the immune-response within the body in the case of diseases other than leukemia, such as a cold[[,]], hence Hence, determination of the white blood cell counts alone may allow false-positive results. Besides, normal white blood cell counts in the peripheral blood range as broad as 4,000-8,000 cell/µL 4,000-8,000 cells/µL, so that false-negative cases are possibly raised. Therefore, a method for diagnosing leukemia with higher accuracy is in need.

Please replace the paragraph on page 2, second full paragraph in the middle, beginning with "[a]s a method for diagnosing recurrence . . . ," with the following paragraph.

As a method for diagnosing recurrence of leukemia, detection by RT-PCR for of the WT-1 gene is mentioned [Clinical Pathology, 48, (underlining in original) 155 (2000), Blood,

84, (underlining in original) 3071 (1994), Japanese Patent No. 3122771]. This diagnostic method is complicated in handling, and requires a special device. As therapies for leukemia, pre-leukemia or aleukemic malignant blood diseases mentioned above, congenital metabolic diseases, solid cancers and the like, the hematopoietic stem cell transplantation therapy is mentioned. Drawbacks associated with the hematopoietic stem cell transplantation therapy include the HLA-type incompatibility between the hematopoietic stem cells of a donor and a patient, insufficient effect of the therapy owing to the physical condition of a patient and to infection, etc., such as non-engraftment of the transplanted hematopoietic stem cells, delayed engraftment of the hematopoietic stem cells, occurrence of graft versus host disease (hereinafter referred to as GVHD). When things turn to the worst, it may proceed to death.

Please replace the paragraph beginning on page 2, last sentence at the bottom, beginning with "[d]elayed engraftment of the hematopoietic stem cells ...," and continuing on page 3, with the following paragraph.

Delayed engraftment of the hematopoietic stem cells can be coped with <u>by the</u> in-vivo administration of G-CSF to promote the engraftment of the hematopoietic stem cells. To cope with GVHD, in-vivo administration of immunosuppressants also suppresses rejection of the transplanted hematopoietic stem cells. However, there is a fear of side effects for either kind of agents, if administered in an excess amount. Therefore, it is crucial for determining the treatment strategy to diagnose or predict the engraftment of the hematopoietic stem cells or the occurrence of GVHD.

Please replace the paragraph on page 3, the second to last full paragraph, towards the bottom, beginning with "[a] method for diagnosing the occurrence. . . ," with the following paragraph.

A method for diagnosing the occurrence of GVHD includes observing skin rush or the like emerging at a recovery phase after transplantation of the hematopoietic stem cells. However, an easy and accurate method for diagnosing the occurrence of GVHD remains unknown. Moreover, any method to predict the occurrence of GVHD prior to its occurrence is unknown either.

Please replace the paragraph on page 3, last full paragraph, beginning with "[h]uman stem cell growth factor . . . ," with the following paragraph.

Human stem cell growth factor (hereinafter abbreviated as SCGF) is a protein comprising an amino acid sequence of SEQ. ID No. 1 or 2 [WO98/08859 WO98/08869, Proc. Natl. Acad. Sci. USA, 94, 7577 (1997), Biochem. Biophys. Res. Comm., 249, (underlining in original) 124 (1998)].

Please replace the first paragraph on page 4, beginning with "[a]s antibodies which recognize SCGF...," with the following paragraph.

As Among antibodies which known to recognize SCGF, known there are a polyclonal antibody antibodies which is are prepared by using SCGF obtained by genetic recombination and a partial peptide of SCGF which consists of 6-25 amino acid residues from the N-terminal N-terminus, as an immunogen immunogens, and monoclonal antibodies which are prepared by using SCGF purified partially from the cell culture supernatant and SCGF obtained by genetic recombination, as an immunogen immunogens [WO98/08859 WO98/08869]. The reference reports that this monoclonal antibody has a neutralizing activity, that a polyclonal antibody, which is prepared by using SCGF obtained by genetic recombination as an immunogen, reacts with SCGF obtained by genetic recombination when subjected to ELISA, and that SCGF obtained by genetic recombination can be detected by western blotting using a polyclonal

antibody prepared by using a partial peptide of SCGF consisting of 6-25 amino acid residues from N-terminal the N-terminus as an immunogen.

V

Please replace the paragraph on page 4, the second full paragraph in the middle, beginning with "[f]urther, there is a report as to anti-SCGF . . . ," with the following paragraph.

Further, there is a report as to anti-SCGF monoclonal KM2142 antibody which is prepared by using a partial peptide of SCGF consisting of 6-25 amino acid residues from the N-terminal N-terminus as an immunogen [The Hematology Journal, 2, (underlining in original) 307 (2001)].

Please replace the paragraph on page 4, last paragraph towards the bottom, beginning with "[i]t is known that the expression levels of SCGF . . . ," with the following paragraph.

It is known that the expression levels of the SCGF gene, as revealed by northern blotting for human normal tissues, are high in the kidney, low in the heart, and nil in the brain, placenta, lung, liver, skeletal muscles and pancreas [Proc. Natl. Acad. Sci. USA, 94, (underlining in original) 7577 (1997)], high in the spleen, thymus, cecum, bone marrows marrow, fetal liver and low in peripheral blood [Biochem. Biophys. Res. Comm., 249, (underlining in original) 124 (1998)]. Also, it is reported that, as a result of in-situ hybridization with normal new-born mice, SCGF is expressed in the bone marrows marrow, proliferating cartilage, and in the proximal of periosteum [The Hematology Journal, 2, (underlining in original) 307 (2001)].

Please replace the paragraph on page 8, towards the bottom, beginning with "[f]ig. 2 shows the results . . . ," and continuing on page 9, with the following paragraph.

Fig. 2 shows the results of SDS-PAGE and western blotting for the purified human SCGF protein. Lanes 1 and 2 shows show SDS-PAGE patterns of a molecular weight marker and the

purified human SCGF protein. Lanes 3, 4 and 5 indicate the results of western blotting for the purified human SCGF protein using KM2142, KM2804 and KM2945, respectively.

1: Lane for a molecular weight marker.

V

- 2. Lane for the purified SCGF which was analyzed and silver-stained.
- 3. Lane subjected to western blotting using the anti-SCGF KM2142 antibody.
- 4. Lane subjected to western blotting using the anti-SCGF KM2804 antibody.
- 5. Lane subjected to western blotting using the anti-SCGF KM2945 antibody.
- A: indicating the molecular weight of SCGF protein.
- B: indicating the molecular weight of the SCGF protein  $\Delta 28$  which lacks the 28th amino acid residue from the N-terminus.
- C: indicating the molecular weight of the SCGF protein  $\Delta 59$  which lacks the 59th amino acid residue from the N-terminal N-terminus.

Please replace the second full paragraph on page 11, towards the middle, beginning with "[a]ny type of leukemia is encompassed . . . ," with the following paragraph.

Any type of leukemia is encompassed in the present invention as long as immature cells, such as hematopoietic cells among the cells of the hematopoietic system, have turned into tumors[[,]]. and the examples Examples include acute lymphocytic leukemia (hereinafter referred to as ALL), acute myeloid leukemia (hereinafter referred to as AML), and chronic myeloid leukemia (hereinafter referred to as CML).

Please replace the third full paragraph on page 11, in the middle, beginning with "[a]ny type of pre-leukemia is emcompassed . . . ," with the following paragraph.

Any type of pre-leukemia is emcompassed encompassed in the present invention as long as mature cells, such as lymphocytes among the cells of the hematopoietic system, have turned into tumors[[,]]. and the examples include An example includes myelodysplastic syndrome (hereinafter referred to as MDS).

Please replace the paragraph on page 11, last paragraph at the bottom, beginning with "SCGF concentrations of in-vivo samples . . . ," with the following paragraph.

V

SCGF concentrations of in-vivo samples of patients with leukemia, pre-leukemia and aleukemic malignant blood diseases are significantly increased compared to those of healthy individuals. A Cut-off cut-off value is therefore applied to the SCGF concentration in quantifying SCGF contained in the in-vivo samples collected, and it can be diagnosed as being leukemia, pre-leukemia or aleukemic malignant blood diseases when a the SCGF concentration exceeds the cut-off value.

Please replace the paragraph on page 13, last paragraph at the bottom, beginning with "[i]n diagnosing whether being leukemia, pre-leukemia...," and continuing on page 14, with the following paragraph.

In diagnosing whether being leukemia, pre-leukemia or aleukemic malignant blood diseases or not, diagnosis can be given at a sensitivity of 89.5% and a specificity of 70% when a cut-off value is set at 15.0 ng/mL, and at a sensitivity of 100% and a specificity of 60% when a cut-off value is set at 13.0 ng/mL. When a cut-off value is set at 18.2 ng/mL which is "mean + 2SD" from the SCGF concentration of healthy individuals, diagnosis can be given at a sensitivity of 89.5% and a specificity of 100%. Moreover, by the use of this cut-off value, whether being leukemia or not can be diagnosed at a sensitivity of 95% and a specificity of 100%, whether being an aleukemic malignant blood disease or not at a sensitivity of 76.9% and a specificity of 100%, and whether being pre-leukemia or not at a sensitivity of 100% and a specificity of 100%.

Please replace the paragraph on page 14, last paragraph at the bottom, beginning with "[a]plastic anemia and myelodysplastic syndrome . . . ," and continuing on page 15, with the following paragraph.

Aplastic anemia and myelodysplastic syndrome have pathologies characterized in that by abnormalities are raised in the counts and morphology of white blood cells in the bone marrows marrow and peripheral blood: Discrimination of the two diseases have has been considered to be difficult.

V

Please replace the paragraph on page 15, first full paragraph, beginning with "SCGF concentration of a myelodysplastic . . . ," with the following paragraph.

The SCGF concentration of a myelodysplastic syndrome patient has been significantly increased compared to that in the blood of a healthy individual, while the blood SCGF concentration of an aplastic anemia patient is comparable to that of a healthy individual. The blood SCGF concentration of a myelodysplastic syndrome patient is significantly higher than that of an aplastic anemia patient, so that measuring the blood SCGF concentrations of patients of the two diseases enables to discriminate discrimination between aplastic anemia and myelodysplastic syndrome.

Please replace the paragraph on page 15, second full paragraph, beginning with "[f]or discriminating between patients of aplastic anemia . . . ," with the following paragraph.

For discriminating between patients of aplastic anemia and of myelodysplastic syndrome, among the patients accompanying abnormality of white blood cells, patients of the two diseases of interest can be discriminated at a sensitivity of 100% and a specificity of 100% by setting a cut-off value (average + 2SD = 16.6 ng/mL) from SCGF concentrations of the aplastic anemia patients and diagnosing on the basis of the cut-off value. Further, by setting the standard value between 15.6 ng/mL and 18.6 ng/mL, patients of aplastic anemia and of myelodysplastic syndrome can be discriminated at a sensitivity of 100% and a specificity of 100%.

Please replace the paragraph on page 16, second full paragraph, beginning with "[w]ith regard to SCGF concentrations . . . ," with the following paragraph.

4

¥

With regard to SCGF concentrations at the pre-conditioning and aplastic phases of the invivo samples of the patients subjected to transplantation of the hematopoeitic stem cells, the concentrations of the in-vivo samples of the patients with delayed engraftment of the hematopoietic stem cells are higher than those of the patients without delayed engraftment of the hematopoietic stem cells. Therefore, the SCGF concentration in each phase is measured[[,]]. then Then the SCGF concentration which is considered as possibly resulting in delayed engraftment of the hematopoietic stem cells is specified as a cut-off value[[,]]. and when When a the SCGF concentration is lower than the cut-off value, it can be diagnosed as free of delayed engraftment. and when When a the SCGF concentration is higher than the cut-off value, it can be diagnosed that delayed engraftment should occur.

Please replace the paragraph on page 18, first full paragraph, beginning with "[e]xamples of molecular-biological assay . . . ," with the following paragraph.

Examples of molecular-biological assays include the RT-PCR method, northern blotting method, in situ hybridization method and the like.

Please replace the paragraph on page 20, first full paragraph, beginning with "[p]rocedure of sandwich assay . . . ," with the following paragraph.

The Procedure procedure of a sandwich assay is described as follows. A second antibody (secondary antibody) is simultaneously or independently reacted with the object substance in a sample together with the primary antibody that were was bound through the antigen-antibody reaction[[,]]. then Then the object substance in the sample is detected or quantified with the use of the same or different antibodies. In many cases, this method comprises in the course of a

measuring operation, a step to wash away unreacted sample components or components of the measuring system in the sample. For example, after the first antibody (primary antibody) is fixed to the solid phase, a sample to be measured is brought into contact with the first sample antibody. Unreacted sample components in the sample are washed and removed from the reaction system[[,]]. then Then the second antibody (secondary antibody) is reacted with the complex of the object substance in the sample and the first antibody that were bound through the antigen-antibody reaction. After components, such as a secondary antibody, that were not involved in the reaction that occurred within the measuring system are washed and removed, the object substance in the sample in the reaction system is detected or quantified.

Y

Please replace the paragraph on page 24, second full paragraph in the middle, beginning with "[a] calibration curve can be obtained . . . ," with the following paragraph.

A calibration curve can be obtained by providing as a standard substance a solution serially diluted to several folds fold which contains a human SCGF protein with a known concentration, and by carrying out [[a]] the sandwich assay described above using in-vivo samples.

Please replace the paragraph on page 24, third full paragraph in the middle, beginning with "[a]s an antibody to SCGF...," with the following paragraph.

As For an antibody to SCGF contained in a diagnostic agent for leukemia, pre-leukemia or aleukemic malignant blood diseases, a diagnostic agent for delayed engraftment of the hematopoietic stem cells after transplantation of the hematopoietic stem cells, and a diagnostic agent for GVHD occurrence, of the present invention, any antibody react reacting with SCGF such as polyclonal antibodies, monoclonal antibodies or antibody fragments may be used, and monoclonal antibodies are preferred.

Please replace the paragraph on page 29, first full paragraph, beginning with "[c]ystein is

added to a partial ...," with the following paragraph.

 $\mathbf{Y}^{n}$ 

Cystein Cysteine is added to a partial peptide on its terminal terminus for the cross-

linking with the protein. When an internal sequence of the protein is selected, the N-terminal N-

terminus of the peptide is acetylated and the C terminal C-terminus is amidated, if necessary.

Please replace the paragraph on page 32, first full paragraph in the middle, beginning

with "[a] part of the culture supernatant . . . ," with the following paragraph.

A part of the culture supernatant is collected after the cultivation and subjected to, for

example, Enzyme-linked an enzyme-linked immunosorbent assay as described below, to select

cells which react to human SCGF but does do not react to antigens free from human SCGF.

Subsequently, cloning is repeated twice using a limiting dilution method [a HT medium (HAT

medium removed of aminopterin) is used for the first time and a normal medium is used for the

second time], and those observed for stable and high antibody titers are selected as the human

SCGF monoclonal antibody-producing hybridoma lines.

Please replace the paragraph on page 34, the third full paragraph in the middle, beginning

with "[t]he following brevity codes represent . . . ," with the following paragraph.

The following brevity codes represent the corresponding amino acids as listed below

unless otherwise stated.

Ala: L-Alanine

Arg: L-Arginine

Cys: L-Cysteine

Gln: L-Glutamine

Glu: L-Glutamic acid

-11-

Glx: L-Glutamic acid

Gly: Glycine

٨,

Leu: L-Leucine

Trp: L-Tryptophan

Please replace the paragraph on page 40, second full paragraph, beginning with "P3-U1, the 8-azaguanine-resistance . . . ," with the following paragraph.

P3-U1, the 8-azaguanine-resistance 8-azaguanine-resistant mouse myeloma cell line, was cultured in a normal medium, and  $2 \times 10^7$  or more cells are were reserved at the time of cell fusion and provided as a parent line for fusing the cells.

Please replace the paragraph on page 41, last paragraph beginning in the middle, beginning with "[t]he hybridoma lines obtained. . . ," and continuing on page 42, with the following paragraph.

The hybridoma lines obtained in Example 1 (6) were intraperitoneally injected to into 8-week-old nude female mice (Balb/c) that had been treated with Pristane at 5-20 x 10<sup>6</sup> cells per mouse. The hybridomas turned into ascites tumor 10 to 21 days after the injection. Ascitic fluid is was collected from the mice accumulating ascitic fluid (1-8 mL/mouse), and the solid was removed by centrifugation (3,000 rpm, 5 min). When IgM was adopted as a monoclonal antibody, salting out was carried out with 50% ammonium sulfate and dialysis was carried out with PBS which had been added 0.5 M sodium chloride[[,]]. The IgM fraction was then collected by passing through a Cellurofine GSL2000 column (SEIKAGAKU CORPORATION) (bed volume of 750 mL) at a flow rate of 15 mL/h to give a purified monoclonal antibody. When IgG was adopted as a monoclonal antibody, purification was carried out by the caprylic

acid precipitation [Antibodies - A Laboratory Manual, Cold Spring Harbor Laboratory (1988)] to give a purified monoclonal antibody.

Please replace the paragraph on page 43, first paragraph, beginning with "[t]he plasmid was introduced into . . . ," with the following paragraph.

The plasmid was introduced into animal cells by an electroporation method according to the method of Miyaji et al. [Miyaji et al., Cytotechnology, 3, (underlining in original) 133-140 (1990)]. 4 ug of pAGE-SCGF-a was introduced into 4 x 10<sup>6</sup> cells of the dhfr gene-deficient CHO cell line [Urlaub and Chasin, Proc. Natl. Acad. Sci. USA, 77, (underlining in original) 4216-4220 (1980)]. These cells were suspended in [[a]] 10 mL MEMa2000-dFCS (5) medium (MEMa2000 medium (GIBCO/BRL) containing 5% dFCS, 1/40 volume of 7.5% NaHCO<sub>3</sub>, 200 mL of 3% glutamine solution (GIBCO/BRL), and 0.5% penicillin/streptomycin solution (GIBCO/BRL, containing 5,000 units/mL penicillin and 5,000 mg/mL streptomycin)], then placed in a 10-cm plate (IWAKI & Co. Ltd.), and cultured in a CO<sub>2</sub> incubator under 37° C for 24 h. Hygromycin (GIBCO/BRL) was added thereto to the final concentration of 0.3 mg/mL, and the cells were further cultured for 1 to 2 weeks. The cells were collected when the transformant cells become confluent, suspended to a in MEMa2000-dFCS (5) medium containing 0.3 mg/mL of hygromycin and 50 nmol/L of methotrexate (MTX) at 1-2 x 10<sup>6</sup> cells/mL, and then pipetted to F75 flasks (Greiner) by 2 mL. After [[the]] cultivation for 1 to 2 weeks, those cells resistant to 50 nmol/L MTX were suspended to a in MEMa2000-dFCS (5) medium containing 0.3 mg/mL hygromycin and 200 nmol/L MTX at 1-2 x 10<sup>5</sup> cells/mL, and pipetted to F75 flasks (Greiner) by 2 mL. After [[the]] cultivation for 1 to 2 weeks, the cells resistant to 200 nmol/L MTX were obtained. These cells resistant to 200 nmol/L MTX were cultured in a 2 L-roller bottle (Greiner) at 37° C and 80 rounds/min by using the medium 1) and medium 2) shown below.

Medium 1) Ex-cell 301 serum-free medium (JRH Biosciences).

Medium 2) Ex-cell 301 serum-free medium containing 10 mg/L aprotinin (Sigma).

Please replace the paragraph on page 51, second full paragraph, beginning with "[w]hen the undenatured human SCGF...," with the following paragraph.

When the undenatured human SCGF protein described in Example 3 was used as an immunogen, any no monoclonal antibody reacting with  $\Delta 59$  was [[not]] obtained. Accordingly, in order to produce a monoclonal antibody reacting with  $\Delta 59$ , a hybridoma was prepared using the SDS-denatured SCGF as an antigen.

Please replace the paragraph on page 54, third paragraph in the middle, beginning with "[a]n anti-SCGF monoclonal KM2142 . . . ," with the following paragraph.

An anti-SCGF monoclonal KM2142 antibody is a hybridoma-derived antibody prepared by using a partial peptide corresponding to the 6-25 residues on the N-terminal N-terminus of the SCGF amino acid sequence shown by SEQ. ID No. 1 (Compound 1) as an antigen. The anti-SCGF monoclonal KM2142 antibody has been shown to possess reactivity to SCGF protein as well. The anti-SCGF monoclonal KM2142 antibody has been further shown to possess reactivity to both human and mouse SCGF proteins.

Please replace the paragraph on page 55, second full paragraph in the middle, beginning with "[l]anes 3, 4 and 5 in Fig. 2 . . . ," with the following paragraph.

Lanes 3, 4 and 5 in Fig. 2 represent the results of western blotting for the purified human SCGF proteins using KM2142, KM2804 and KM2945, respectively. Although KM2804 did not possess reactivity to Δ59 which is a SCGF protein lacking the 59th residue on from the N-terminal N-terminus, it possessed reactivity to Δ28 which is a SCGF protein lacking the 28th

residue on <u>from</u> the N-terminal N-terminus. KM2945 possessed reactivity to both the full-length and deletion-type SCGFs.

Please replace the first full paragraph on page 56, beginning with "[a]n anti-human SCGF monoclonal KM2804 . . . ," with the following paragraph.

An anti-human SCGF monoclonal KM2804 antibody obtained in Example 3 was pipetted to a 96-well EIA plate (Greiner) at 5 µg/mL and 50 µL/well, and the plate was left overnight at 4°C to allow the antibody to be adsorbed. After washing the plate, 1% BSA-PBS was added at 100 uL/well, which was subjected to reaction for 1 h at room temperature to block any remaining active group. The 1% BSA-PBS was discarded, then the CHO cell-expressing human SCGF protein obtained in Example 2 (4), which was diluted with a serum dilution solution (Kyowa Medex Co., Ltd.) 14 times in a two-fold dilution line starting from 17.5 ng/mL, was pipetted at 50 μL/well and allowed to react for 2 h at room temperature. After washing with Tween-PBS, the biotin-labeled KM2142 obtained in the above (diluted to 0.2 µg/mL with BSA-PBS) was added at 50 µL/well, which was subjected to reaction for 2 h at room temperature. After washing with Tween-PBS, alkaline phosphatase-labeled avidin (Zymed Laboratories, Inc.) was further added at x32,000 dilution at 50 µL/well, which was subjected to reaction for 1 h at room temperature. After washing with Tween-PBS, AmpliQ (DAKO Corp.) was added thereto for color development, and the absorbance at OD490 nm was measured with a plate reader (E-max; Molecular Devices). The results revealed that the determination system of the present invention enabled to quantify quantification of the human SCGF protein in a range of 0.04-2.0 ng/mL as shown in Fig. 6.

Please replace the first full paragraph on page 57, beginning with "[t]he SCGF concentrations of sera . . . ," with the following paragraph.

The SCGF concentrations of sera from patients with informed consent <u>and</u> who suffer leukemia, pre-leukemia or aleukemic malignant blood diseases were determined by the method of Example 6. Ten healthy individuals, both men and women, who <u>exhibit exhibited</u> normal test levels in the blood cell test <del>were</del> served as control examples, and their serum SCGF concentrations were also determined. The results are shown in Fig. 7.

Please replace the first full paragraph on page 58, beginning with "[i]t was also demonstrated that . . . ," with the following paragraph.

It was also demonstrated that patients of with leukemia, pre-leukemia or aleukemic malignant blood diseases can be detected at a high senseitivity sensitivity with the use of a cut-off value set down from the values for healthy individuals (Table 4). On the other hand, in spite of being a blood disease as well, no significant difference was observed between the values for patients of with aplastic anemia (AA) and healthy individuals, and this disease was not detectable even with the use of a cut-off value.

Please replace the paragraph on page 59, second full paragraph, beginning with "[a]mong the patients who underwent . . . ," with the following paragraph.

Among the patients who underwent transplantation of the hematopoietic stem cells, cases with the occurrence of GVHD exhibited significantly higher serum SCGF concentrations in aplastic and recovery phases compared to those in the cases without occurrence of the disease.

Thus, the occurrence of GVHD was able to diagnose diagnosed by measuring the serum SCGF concentration.

Please replace the paragraph on page 61, first full paragraph, beginning with "[p]eripheral blood cells of various . . . ," with the following paragraph.

Peripheral blood cells of various leukemia patients with informed consent were subjected to treatment with Rneasy Mini Kit (Qiagen) according to the protocol to extract total RNA.

Then 1 µg of the total RNA was treated with DNaseI (GIBCO), reversely transcribed using SuperScript First-Strand Synthesis System for RT-PCR (GIBCO), and thus First-Strand DNA was prepared. The First-Strand DNA thus prepared was subjected to detection of the human G3PDH and SCGF genes using Taq Polymerase (TaKaRa), the prepared First-Strand DNA as a template, and oligo DNA having base sequences of SEQ. ID Nos. 8 and 9, and SEP SEQ ID Nos. 10 and 11 as primers. As a result, although [[the]] SCGF expression was not detected for a single healthy individual, [[the]] SCGF expression was detected in 1 out of 2 cases for acute lymphocytic leukemia (ALL) and 2 out of 2 cases for acute myeloid leukemia (AML) under the conditions where detected levels of G3PDH was were almost equal.